REMARKS

Applicants elect Group I. (Claims 1-4 and 22-23) without traverse. These claims are directed to a method of modulating movement of a cell within or to the skin of a mammal, said method comprising administering to said mammal an effective amount of an antagonist of CTACK.

Applicants also select one species from type of cell movement group (a. within said skin), one species from the cell type group (d. a CLA+ cell), one species from the agent of combination administration group (m. an antibody that neutralizes CTACK) and one species from the antagonist group (bb. an antibiotic).

STATUS OF CLAIMS

Claims 5-21 are cancelled as they are drawn to non-elected inventions. As a result of the above claim cancellations only claims 1-4 and 22-23 remain pending.

Applicants have amended claims 1-4 and 22-23 for greater clarity. Support for these claim amendments can be found in the specification and the originally filed claims.

No new matter has been added by these claim amendments.

Applicants attach Appendix A with the newly revised claims, primarily for the Examiner's convenience.

In addition, Applicants attach Appendix B a marked-up version of the changes made to the claims by the current amendment entitled "VERSION WITH MARKINGS TO SHOW CHANGES MADE."

CONCLUSION

Applicants reserve the right to file subsequent applications claiming the non-elected subject matter and do not waive any of their rights or abandon any non-elected subject matter. It is believed that the foregoing amendment places this application now in condition for early action. Therefore, early and favorable action allowing pending claims 1-4 and 22-23 is respectfully solicited.

Respectfully submitted,

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SEP 2 3 2002 S WEAT IS SEE AIMED IS:

DX0882XK US REVISED CLAIMS – SEPTEMBER 2002

- 1. A method of modulating movement of a cell within or to the skin of a mammal, said method comprising administering to said mammal an effective amount of an antagonist of CTACK.
- 2. The method of Claim 1, wherein said modulating is blocking and said administering is an antagonist of CTACK.

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3. The method of Claim 2, wherein:

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Page 1 of 1

- a) said movement is within said skin;
- b) said administering is local, systemic, topical, subcutaneous, intradermal, or transdermal;
- c) said administering is an antagonist of CTACK;
- d) said cell is a CLA+ cell; or
- e) said cell moves into the dermis and/or epidermis layers of said skin.
- 4. The method of Claim 2, wherein:
 - a) said antagonist is an antibody which neutralizes CTACK;
 - b) said mammal is subject to a transplant or skin graft;
 - c) said antagonist is administered in combination with an antibiotic.
- 22. A method of treating a patient suffering from a skin disorder comprising administering an effective amount of an antagonist against CTACK.
- 23. The method of Claim 22, wherein the antagonist is an antibody.

Applicant: Wang *et al.*; Serial No.: 09/898,751; Filed: July 2, 2001

VERSION WITH MARKINGS TO SHOW CHANGES MADE

- 1. A method of modulating movement of a cell within or to the skin of a mammal, said method comprising administering to said mammal an effective amount of:
 - a) an antagonist of CTACK:
 - b) an agonist of CTACK:
 - e) an antagonist of Victor
 - d) an agonist of Vic.
- 2. The method of Claim 1, wherein said modulating is blocking and said administering is an antagonist of CTACK or Vic.
- 3. The method of Claim 2, wherein:
 - a) said movement is:
 - ++ within said skin:
 - ii) chemotactic: or
 - iii) chemokinetic:
 - b) said administering is local, systemic, topical, subcutaneous, intradermal, or transdermal;
 - c) said administering is an antagonist of CTACK or Vie;
 - d) said cell is:

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i-a CLA+ cell;
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ii) a l cell:

iii) a dendritic cell; or

iv) a dendritic cell precursor:

v) - a dermal fibroblast cell:

vi) a dermal endothelial cell; or

vii)a melanoevte: or

- e) said cell moves into the dermis and/or epidermis layers of said skin.
- 4. The method of Claim 2, wherein:
 - a) said antagonist is selected from:

i) a mutein of natural CTACK or Vic:

ii) an antibody which neutralizes CTACK or Vic. or

iii) an antibody which blocks GPR2 ligand binding;

b) said mammal is subject to a transplant or skin graft;

Appendix B

	c) said antagonist is administered in combination with an antibiotic, analgesic, immune
	suppressive therapeutic, anti-inflammatory drug, growth factor, or immune
	adjuvant .
<u> 5</u>	————The method of Claim 1, wherein said modulating is attracting and said
admi	nistering is an agonist of CTACK or Vie.
()	The method of Claim 5, wherein:
	a) said movement is:
	i) within said skin:
	ii) chemotactic: or
	iii) chemokinetic:
	b) said administering is local, topical, subcutaneous, intradermal, or transdermal;
	e) said administering is a CTACK or Vic ligand:
	d) said cell is:
	i)—a-CLA+-vell:
	ii) a T cell:
	iii) a dendritic cell: or
	iv) a dendritie cell precursor:
	v) a dermal fibroblast cell:
	vi) a dermal endothelial cell; or
	vii)a melanocyte: or
	e) said cell moves into the dermis and or epidermis layers of said skin.
7	
	a) said agonist is selected from:
	i) CTACK or Vie: or
	ii) a GPR2 ligand:
	b) said mammal is subject to a cutaneous lesion, tumor or viral, microbial, or parasitic
	infection:
	e)-said agonist is administered in combination with an antibiotic, analgesic, immune
	suppressive therapeutic, anti-inflammatory drug, growth factor, or immune
	adjuvant .
8	——————————————————————————————————————
adju	vant.

9 A method of purifying a population of cells, said method comprising contacting
said cells with CTACK or Vic. thereby resulting in the identification of cells expressing a
receptor for said CTACK or Vic.
10 The method of Claim 9, wherein said contacting results in specific movement of
said cells to a site for purification.
11. The method of Claim 9, wherein said movement is through pores of a membrane.
12 A method of producing a ligand:receptor complex, comprising contacting:
a) a mammalian CTACK with a GPR2 receptor; or
b) a mammalian Vie with a GPR2 receptor;
wherein at least one of said ligand or receptor is recombinant or purified, thereby allowing said
complex to form.
13. — The method of Claim 12, wherein:
a) said complex results in a Ca++ flux:
b) said GPR2 receptor is on a cell:
c) said complex formation results in a physiological change in the cell expressing said
GPR2 receptor:
d) said contacting is in combination with H2 and or interferon-α; or
e) said contacting allows quantitative detection of said ligand.
14 A method of modulating physiology or development of a GPR2 expressing cell
comprising contacting said cell to an agonist or antagonist of a mammalian Vic or CTACK.
wherein one of said GPR2 receptor or said agonist or antagonist is recombinant or purified.
15 The method of Claim 14. wherein:
A) said antagonist is:
1) an antibody which:
a) neutralizes said mammalian Vic:
b) neutralizes said mammalian CTACK; or
c) blocks ligand binding by GPR2; or
2) a mutein of said Vic or CTACK; or
B) said physiology is selected from:

Appendix B
Applicant: Wang et al.; Serial No.: 09/898,751; Filed: July 2, 2001

	1)-a cellular calcium flux :
	2) a chemoattractant response:
	3) a cellular morphology modification response:
	4) phosphoinositide lipid turnover: or
	5) an antiviral response:
16	- The method of Claim 15, wherein:
a) saic	l antagonist is an antibody and said physiology is a chemoattractant response; or
b) -saic	d modulating is blocking, and said physiology is an inflammatory response.
interaction, sa	-A-method of testing a compound for ability to affect GPR2 receptor-ligand id method comprising comparing the interaction of GPR2 with Vic or CTACK in and absence of said compound.
18. Vic. or CTAC	-The method of Claim 17, wherein said compound is an antibody against GPR2. K.
19	A primate GPR2, comprising sequence of MGTFVLEQ (see SEQ ID NO)-2).
20.	-A nucleic acid encoding said GPR2 of Claim 19.
21.	An antibody which binds selectively to MGTEVLEQ (see SEQ ID NO; 2).
22. administering	A method of treating a patient suffering from a skin disorder comprising an effective amount of an antagonist against GPR2. Vie. or CTACK.
23.	The method of Claim 22, wherein the antagonist is an antibody.

Appendix B
Applicant: Wang et al.; Serial No.: 09/898,751; Filed: July 2, 2001